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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/526,372	03/03/2005	IWAO OHIZUMI	1254-0274PUS1	3366

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BIRCH STEWART KOLASCH & BIRCH
PO BOX 747
FALLS CHURCH, VA 22040-0747

EXAMINER	
HADDAD, MAHER M	
ART UNIT	PAPER NUMBER
1644	

SHORTENED STATUTORY PERIOD OF RESPONSE	NOTIFICATION DATE	DELIVERY MODE
3 MONTHS	02/09/2007	ELECTRONIC

Please find below and/or attached an Office communication concerning this application or proceeding.

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

Notice of this Office communication was sent electronically on the above-indicated "Notification Date" and has a shortened statutory period for reply of 3 MONTHS from 02/09/2007.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

mailroom@bskb.com

Office Action Summary	Application No.	Applicant(s)	
	10/526,372	OKUMURA, HIROSHI	
	Examiner	Art Unit	
	Maher M. Haddad	1644	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☐ Responsive to communication(s) filed on _____.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) 1-12 is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) 1-12 is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date 3/3/05 & 2/08/06.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☒ Other: Attachment I.

DETAILED ACTION

1. Claims 1-12 are pending and under examination in the instant application.

2. Applicant's IDS, filed 3/3/05 and 2/8/06, is acknowledged.

3. The following is a quotation of the second paragraph of 35 U.S.C. 112.

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

4. Claims 10-12 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

A. Claims 10-12 are indefinite because they recite amino acid sequence homology without providing a SEQ ID NO reference, it is unclear how the skilled artisan would find the correspondent of those amino acids. Finally, claims 11-12 recite "sequence homology is 90%/94% or higher", it is indefinite to compare amino acids between molecules without structural features for the comparison.

5. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless --

(a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

6. Claims 7-10 are rejected under 35 U.S.C. 102(b) as being anticipated by EP 0 872 488 A1 (IDS ref. No. BA).

The EP '488 publication teach a method for producing an anti-Fas ligand antibody comprising a MPL. lpr/lpr mouse (nonhuman) animal with FAS function defects with an Fas ligand-expressed COS cells as an antigen (see p. 7, line 5-55 in particular). Claim 10 is included because the mouse Fas ligand is highly homologous to the human, 85%.

The reference teachings anticipate the claimed invention.

7. Claims 7-12 are rejected under 35 U.S.C. 102(b) as being anticipated by U.S. Pat. No. 6,235,714.

The `714 patent teaches six MRL/lpr mice were hyperimmunized with target antigen such as EGFR, TNFα, IL-1β among others (see fig. 19 and col., 8, under selection and preparation of CRAAs in particular) to drive the immune system to generate catalytic antibodies. Blood will be obtained from the retro-orbital plexus at ten day intervals (see col., 14, under immunization, col., 43, lines 56-66 in particular). Claims 10-12 are included because the target antigens listed in fig. 19, the antigen protein exhibits high amino acid sequence homology in a human and mouse, wherein the amino acid sequence homology is 90% or 94 % or higher in the absence of evidence to the contrary.

The reference teachings anticipate the claimed invention.

8. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(f) or (g) prior art under 35 U.S.C. 103(a).

9. Claims 7 and 10-12 are rejected under 35 U.S.C. 103(a) as being unpatentable over EP 0 872 488 A1 (IDS ref. No. BA) in view of Fu and Storb (Science, 297:2006-2008, 2002).

The teachings of EP 0 872 488 publication have been discussed, supra.

The claimed invention differs from the reference teachings only by the recitation that the antigen protein exhibits high amino acid sequence homology in a human and a mouse in claim 10, wherein the amino acid sequence homology is 90%/94% or higher in claims 11 and 12.

Fu and Storb teach autoreactive B cells that produce antibody against self-antigen are normally deleted through the Fas receptor/Fas ligand-mediated pathway of apoptosis. However, in mice deficient in either Fas receptor or Fas ligand, autoreactive B cells cannot be deleted. They do not accumulate in follicles but instead became trapped in side the T cell zone of lymphoid tissues, where they continue to proliferate and undergo somatic hypermutation, producing more autoantigody against self-antigen (see page 2007, col., 2, Figure legend). In MLR.Fas^{lpr} mice, autoreactive B cells expressing antiself antibodies spontaneously accumulated in the T cell-rich

zone at the red pulp border of lymphoid tissues. The B lymphocytes underwent somatic hypermutation in this zone (page 2007, 1st col., 1st ¶ in particular).

It would have been obvious to one of ordinary skill in the art at the time the invention was made to substitute Fas ligand taught by the EP `488 publication with the antigen protein which exhibits high amino acid sequence homology in a human and a mouse (self antigens) taught by Fu and Storb in a method for producing an antibody taught by the `488 publication.

One of ordinary skill in the art at the time the invention was made would have been motivated to do so because in mice deficient in either Fas receptor or Fas ligand, autoreactive B cells cannot be deleted. They do not accumulate in follicles but instead became trapped in side the T cell zone of lymphoid tissues, where they continue to proliferate and undergo somatic hypermutation, producing more autoantibody against self-antigen.

From the combined teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention. Therefore, the invention as a whole was *prima facie* obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references, especially in the absence of evidence to the contrary.

10. Claims 1-5 are rejected under 35 U.S.C. 103(a) as being unpatentable over EP 0 872 488 A1 (IDS ref. No. BA) in view of Fu and Storb (Science, 297:2006-2008, 2002) as applied to claims 7 and 10-12 and further in view of Veugelers et al (J BC 274(33):26968-26977, 1999).

The teachings of EP 0 872 488 publication and Fu and Storb reference have been discussed, supra.

The claimed invention differs from the reference teachings only by the recitation that the antigen is a glypican protein.

However, Veugelers et al teach that Glypican-6 as a new member of the glypican family of cell surface heparin sulfate proteoglcans that shows a highly distinctive and regulated developmental expression (see last ¶). Veugelers et al teach that the human and mouse forms of glypican-6 are highly similar in structure with 96% identity (see page 26975, 1st col., 1st ¶ in particular). Finally, for the identification of glypican-6 as a heparin sulfate proteoglycan, Veugelers et al used an antibody specific for native heparin sulfate (10E4) and antibody specific for Δ-HS (3G10), a neo-epitope that includes the Δ-glucuronate generated by the digestion of heparin sulfate by heparitinase (see page 26973, 1st col., Under identification ..., in particular).

It would have been obvious to one of ordinary skill in the art at the time the invention was made to target the glypican-6 protein taught by Veugelers et al as the antigen protein which exhibits high amino acid sequence homology in a human and a mouse (i.e., 96% identity for the human

and 100% identity for the mouse (self-antigen)) in a method for producing an antibody taught by the '488 publication.

Given the teachings of Fu and Storb reference that in mice deficient in either Fas receptor or Fas ligand, autoreactive B cells cannot be deleted. They do not accumulate in follicles but instead became trapped in side the T cell zone of lymphoid tissues, where they continue to proliferate and undergo somatic hypermutation, producing more autoantigody against self-antigen, one of ordinary skill in the art at the time the invention was made would have been motivated to raise antibody against GPC-6 protein to study the highly distinctive and regulated developmental expression or to specifically identify the GPC-6 protein with anti-GPC-6 antibody.

From the combined teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention. Therefore, the invention as a whole was *prima facie* obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references, especially in the absence of evidence to the contrary.

11. Claims 1-6 are rejected under 35 U.S.C. 103(a) as being unpatentable over EP 0 872 488 A1 (IDS ref. No. BA) in view of Fu and Storb (Science, 297:2006-2008, 2002) as applied to claims 7 and 10-12 and further in view of Veugelers et al (Trends in Glycosience and Glycotechnology, 10(52):145-152, 1998).

The teachings of EP 0 872 488 publication and Fu and Storb reference have been discussed, supra.

The claimed invention differs from the reference teachings only by the recitation that the antigen is a glypican 3 protein.

However, Veugelers et al teach that all glypicans share a conserved amino acid sequence motif that includes a characteristic pattern of 14 cystein residues. Additional similarities include the overall sizes of the proteins, the presence of N-terminal and C-terminal signal peptide-like sequences and glycosaminoglycan-attachment consensus sequences that occur towards the C-termini of the proteins. (see page 145, under Glypican Structures in particular). Further, Veugelers et al teach Further teaches that in general, there is a high degree of conservation between the human and rodent forms of the glypicans (approximately 90% at the amino acid level) (See Table II legend in particular). Finally, Veugelers et al teach that GPC3 is identified as a GPI-anchored heparin sulfate proteoglycan of the glypican family, with a core protein Mr of 69 kd (see Page 150, under OCI-5 in particular). Further, an amino acid sequence alignment of the human GPC3 with mouse GPC3 provides a 94.2% amino acid sequence identity (see attached sequence alignment in particular).

It would have been obvious to one of ordinary skill in the art at the time the invention was made to target the glypican-3 protein taught by Veugelers et al as the antigen protein which exhibits high degree of conservation (~90%) between the human and rodent forms of glypicans (e.g.,

94% identity for the human and mouse (self-antigen)) in a method for producing an antibody taught by the `488 publication.

Given the teachings of Fu and Storb reference that in mice deficient in either Fas receptor or Fas ligand, autoreactive B cells cannot be deleted. They do not accumulate in follicles but instead became trapped in side the T cell zone of lymphoid tissues, where they continue to proliferate and undergo somatic hypermutation, producing more autoantibody against self-antigen, one of ordinary skill in the art at the time the invention was made would have been motivated to raise antibody against GPC-3 protein to study the highly distinctive and regulated developmental expression or to specifically identify the GPC-3 protein with anti-GPC-3 antibody.

From the combined teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention. Therefore, the invention as a whole was *prima facie* obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references, especially in the absence of evidence to the contrary.

12. No claim is allowed.

13. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Maher Haddad whose telephone number is (571) 272-0845. The examiner can normally be reached Monday through Friday from 7:30 am to 4:00 pm. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Christina Chan can be reached on (571) 272-0841. The fax number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

January 30, 2007

Maher Haddad

Maher Haddad, Ph.D.
Primary Examiner
Technology Center 1600

Attachment I

<!--StartFragment-->RESULT 7
Q3TWB2_MOUSE
ID   Q3TWB2_MOUSE   PRELIMINARY;   PRT;   579 AA.
AC   Q3TWB2;
DT   11-OCT-2005, integrated into UniProtKB/TrEMBL.
DT   11-OCT-2005, sequence version 1.
DT   27-JUN-2006, entry version 9.
DE   Osteoclast-like cell cDNA, RIKEN full-length enriched library,
DE   clone:I420030H13 product:glypican 3, full insert sequence (17 days
DE   pregnant adult female amnion cDNA, RIKEN full-length enriched library,
DE   clone:I920011I15 product:glypican 3, full insert sequence) (13 days
DE   embryo liver cDNA, RIKEN full-length enriched library,
DE   clone:I920043N06 product:glypican 3, full insert sequence).
GN   Name=Gpc3;
OS   Mus musculus (Mouse).
OC   Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
OC   Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Sciurognathi;
OC   Muroidea; Muridae; Murinae; Mus.
OX   NCBI_TaxID=10090;
RN   [1]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=C57BL/6J; TISSUE=Amnion, and Liver;
RX   MEDLINE=99279253; PubMed=10349636; DOI=10.1016/S0076-6879(99)03004-9;
RA   Carninci P., Hayashizaki Y.;
RT   "High-efficiency full-length cDNA cloning.";
RL   Methods Enzymol. 303:19-44(1999).
RN   [2]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=C57BL/6J; TISSUE=Amnion, and Liver;
RX   PubMed=16141072; DOI=10.1126/science.1112014;
RA   Carninci P., Kasukawa T., Katayama S., Gough J., Frith M.C., Maeda N.,
RA   Oyama R., Ravasi T., Lenhard B., Wells C., Kodzius R., Shimokawa K.,
RA   Bajic V.B., Brenner S.E., Batalov S., Forrest A.R., Zavolan M.,
RA   Davis M.J., Wilming L.G., Aidinis V., Allen J.E.,
RA   Ambesi-Impiombato A., Apweiler R., Aturaliya R.N., Bailey T.L.,
RA   Bansal M., Baxter L., Beisel K.W., Bersano T., Bono H., Chalk A.M.,
RA   Chiu K.P., Choudhary V., Christoffels A., Clutterbuck D.R.,
RA   Crowe M.L., Dalla E., Dalrymple B.P., de Bono B., Della Gatta G.,
RA   di Bernardo D., Down T., Engstrom P., Fagiolini M., Faulkner G.,
RA   Fletcher C.F., Fukushima T., Furuno M., Futaki S., Gariboldi M.,
RA   Georgii-Hemming P., Gingeras T.R., Gojobori T., Green R.E.,
RA   Gustincich S., Harbers M., Hayashi Y., Hensch T.K., Hirokawa N.,
RA   Hill D., Huminiecki L., Iacono M., Ikeo K., Iwama A., Ishikawa T.,
RA   Jakt M., Kanapin A., Katoh M., Kawasawa Y., Kelso J., Kitamura H.,
RA   Kitano H., Kollias G., Krishnan S.P., Kruger A., Kummerfeld S.K.,
RA   Kurochkin I.V., Lareau L.F., Lazarevic D., Lipovich L., Liu J.,
RA   Liuni S., McWilliam S., Madan Babu M., Madera M., Marchionni L.,
RA   Matsuda H., Matsuzawa S., Miki H., Mignone F., Miyake S., Morris K.,
RA   Mottagui-Tabar S., Mulder N., Nakano N., Nakauchi H., Ng P.,
RA   Nilsson R., Nishiguchi S., Nishikawa S., Nori F., Ohara O.,
RA   Okazaki Y., Orlando V., Pang K.C., Pavan W.J., Pavesi G., Pesole G.,
RA   Petrovsky N., Piazza S., Reed J., Reid J.F., Ring B.Z., Ringwald M.,
RA   Rost B., Ruan Y., Salzberg S.L., Sandelin A., Schneider C.,
RA   Schonbach C., Sekiguchi K., Semple C.A., Seno S., Sessa L., Sheng Y.,
RA   Shibata Y., Shimada H., Shimada K., Silva D., Sinclair B.,
RA   Sperling S., Stupka E., Sugiura K., Sultana R., Takenaka Y., Taki K.,
RA   Tammoja K., Tan S.L., Tang S., Taylor M.S., Tegner J., Teichmann S.A.,
RA   Ueda H.R., van Nimwegen E., Verardo R., Wei C.L., Yagi K.,
RA   Yamanishi H., Zabarovsky E., Zhu S., Zimmer A., Hide W., Bult C.,
RA   Grimmond S.M., Teasdale R.D., Liu E.T., Brusic V., Quackenbush J.,
RA   Wahlestedt C., Mattick J.S., Hume D.A., Kai C., Sasaki D., Tomaru Y.,

RA Fukuda S., Kanamori-Katayama M., Suzuki M., Aoki J., Arakawa T.,
RA Iida J., Imamura K., Itoh M., Kato T., Kawaji H., Kawagashira N.,
RA Kawashima T., Kojima M., Kondo S., Konno H., Nakano K., Ninomiya N.,
RA Nishio T., Okada M., Plessy C., Shibata K., Shiraki T., Suzuki S.,
RA Tagami M., Waki K., Watahiki A., Okamura-Oho Y., Suzuki H., Kawai J.,
RA Hayashizaki Y.;
RT "The transcriptional landscape of the mammalian genome.";
RL Science 309:1559-1563(2005).
RN [3]
RP NUCLEOTIDE SEQUENCE.
RC STRAIN=C57BL/6J; TISSUE=Amnion, and Liver;
RX PubMed=16141073; DOI=10.1126/science.1112009;
RG RIKEN Genome Exploration Research Group, and Genome Science Group
RG (Genome Network Core Team) and the FANTOM Consortium;
RT "Antisense Transcription in the Mammalian Transcriptome.";
RL Science 309:1564-1566(2005).
RN [4]
RP NUCLEOTIDE SEQUENCE.
RC STRAIN=C57BL/6J; TISSUE=Amnion, and Liver;
RX MEDLINE=22354683; PubMed=12466851; DOI=10.1038/nature01266;
RA Okazaki Y., Furuno M., Kasukawa T., Adachi J., Bono H., Kondo S.,
RA Nikaido I., Osato N., Saito R., Suzuki H., Yamanaka I., Kiyosawa H.,
RA Yagi K., Tomaru Y., Hasegawa Y., Nogami A., Schonbach C., Gojobori T.,
RA Baldarelli R., Hill D.P., Bult C., Hume D.A., Quackenbush J.,
RA Schriml L.M., Kanapin A., Matsuda H., Batalov S., Beisel K.W.,
RA Blake J.A., Bradt D., Brusic V., Chothia C., Corbani L.E., Cousins S.,
RA Dalla E., Dragani T.A., Fletcher C.F., Forrest A., Frazer K.S.,
RA Gaasterland T., Gariboldi M., Gissi C., Godzik A., Gough J.,
RA Grimmond S., Gustincich S., Hirokawa N., Jackson I.J., Jarvis E.D.,
RA Kanai A., Kawaji H., Kawasawa Y., Kedzierski R.M., King B.L.,
RA Konagaya A., Kurochkin I.V., Lee Y., Lenhard B., Lyons P.A.,
RA Maglott D.R., Maltais L., Marchionni L., McKenzie L., Miki H.,
RA Nagashima T., Numata K., Okido T., Pavan W.J., Pertea G., Pesole G.,
RA Petrovsky N., Pillai R., Pontius J.U., Qi D., Ramachandran S.,
RA Ravasi T., Reed J.C., Reed D.J., Reid J., Ring B.Z., Ringwald M.,
RA Sandelin A., Schneider C., Semple C.A., Setou M., Shimada K.,
RA Sultana R., Takenaka Y., Taylor M.S., Teasdale R.D., Tomita M.,
RA Verardo R., Wagner L., Wahlestedt C., Wang Y., Watanabe Y., Wells C.,
RA Wilming L.G., Wynshaw-Boris A., Yanagisawa M., Yang I., Yang L.,
RA Yuan Z., Zavolan M., Zhu Y., Zimmer A., Carninci P., Hayatsu N.,
RA Hirozane-Kishikawa T., Konno H., Nakamura M., Sakazume N., Sato K.,
RA Shiraki T., Waki K., Kawai J., Aizawa K., Arakawa T., Fukuda S.,
RA Hara A., Hashizume W., Imotani K., Ishii Y., Itoh M., Kagawa I.,
RA Miyazaki A., Sakai K., Sasaki D., Shibata K., Shinagawa A.,
RA Yasunishi A., Yoshino M., Waterston R., Lander E.S., Rogers J.,
RA Birney E., Hayashizaki Y.;
RT "Analysis of the mouse transcriptome based on functional annotation of
RT 60,770 full-length cDNAs.";
RL Nature 420:563-573(2002).
RN [5]
RP NUCLEOTIDE SEQUENCE.
RC STRAIN=C57BL/6J; TISSUE=Amnion, and Liver;
RX MEDLINE=21085660; PubMed=11217851; DOI=10.1038/35055500;
RA Kawai J., Shinagawa A., Shibata K., Yoshino M., Itoh M., Ishii Y.,
RA Arakawa T., Hara A., Fukunishi Y., Konno H., Adachi J., Fukuda S.,
RA Aizawa K., Izawa M., Nishi K., Kiyosawa H., Kondo S., Yamanaka I.,
RA Saito T., Okazaki Y., Gojobori T., Bono H., Kasukawa T., Saito R.,
RA Kadota K., Matsuda H.A., Ashburner M., Batalov S., Casavant T.,
RA Fleischmann W., Gaasterland T., Gissi C., King B., Kochiwa H.,
RA Kuehl P., Lewis S., Matsuo Y., Nikaido I., Pesole G., Quackenbush J.,
RA Schriml L.M., Staubli F., Suzuki R., Tomita M., Wagner L., Washio T.,

RA Sakai K., Okido T., Furuno M., Aono H., Baldarelli R., Barsh G.,
RA Blake J., Boffelli D., Bojunga N., Carninci P., de Bonaldo M.F.,
RA Brownstein M.J., Bult C., Fletcher C., Fujita M., Gariboldi M.,
RA Gustincich S., Hill D., Hofmann M., Hume D.A., Kamiya M., Lee N.H.,
RA Lyons P., Marchionni L., Mashima J., Mazzarelli J., Mombaerts P.,
RA Nordone P., Ring B., Ringwald M., Rodriguez I., Sakamoto N.,
RA Sasaki H., Sato K., Schoenbach C., Seya T., Shibata Y., Storch K.-F.,
RA Suzuki H., Toyo-oka K., Wang K.H., Weitz C., Whittaker C., Wilming L.,
RA Wynshaw-Boris A., Yoshida K., Hasegawa Y., Kawaji H., Kohtsuki S.,
RA Hayashizaki Y.;
RT "Functional annotation of a full-length mouse cDNA collection.";
RL Nature 409:685-690(2001).
RN [6]
RP NUCLEOTIDE SEQUENCE.
RC STRAIN=C57BL/6J; TISSUE=Amnion, and Liver;
RX MEDLINE=20499374; PubMed=11042159; DOI=10.1101/gr.145100;
RA Carninci P., Shibata Y., Hayatsu N., Sugahara Y., Shibata K., Itoh M.,
RA Konno H., Okazaki Y., Muramatsu M., Hayashizaki Y.;
RT "Normalization and subtraction of cap-trapper-selected cDNAs to
RT prepare full-length cDNA libraries for rapid discovery of new genes.";
RL Genome Res. 10:1617-1630(2000).
RN [7]
RP NUCLEOTIDE SEQUENCE.
RC STRAIN=C57BL/6J; TISSUE=Amnion, and Liver;
RX MEDLINE=20530913; PubMed=11076861; DOI=10.1101/gr.152600;
RA Shibata K., Itoh M., Aizawa K., Nagaoka S., Sasaki N., Carninci P.,
RA Konno H., Akiyama J., Nishi K., Kitsunai T., Tashiro H., Itoh M.,
RA Sumi N., Ishii Y., Nakamura S., Hazama M., Nishine T., Harada A.,
RA Yamamoto R., Matsumoto H., Sakaguchi S., Ikegami T., Kashiwagi K.,
RA Fujiwake S., Inoue K., Togawa Y., Izawa M., Ohara E., Watahiki M.,
RA Yoneda Y., Ishikawa T., Ozawa K., Tanaka T., Matsuura S., Kawai J.,
RA Okazaki Y., Muramatsu M., Inoue Y., Kira A., Hayashizaki Y.;
RT "RIKEN integrated sequence analysis (RISA) system-384-format
RT sequencing pipeline with 384 multicapillary sequencer.";
RL Genome Res. 10:1757-1771(2000).
RN [8]
RP NUCLEOTIDE SEQUENCE.
RC STRAIN=C57BL/6J; TISSUE=Liver;
RA Arakawa T., Carninci P., Fukuda S., Hashizume W., Hayashida K.,
RA Hori F., Iida J., Imamura K., Imotani K., Itoh M., Kanagawa S.,
RA Kawai J., Kojima M., Konno H., Murata M., Nakamura M., Ninomiya N.,
RA Nishiyori H., Nomura K., Ohno M., Sakazume N., Sano H., Sasaki D.,
RA Shibata K., Shiraki T., Tagami M., Tagami Y., Waki K., Watahiki A.,
RA Muramatsu M., Hayashizaki Y.;
RL Submitted (MAR-2004) to the EMBL/GenBank/DDBJ databases.
RN [9]
RP NUCLEOTIDE SEQUENCE.
RC STRAIN=C57BL/6J; TISSUE=Amnion;
RA Arakawa T., Carninci P., Fukuda S., Hashizume W., Hayashida K.,
RA Hori F., Iida J., Imamura K., Imotani K., Itoh M., Kanagawa S.,
RA Kawai J., Kojima M., Konno H., Murata M., Nakamura M., Ninomiya N.,
RA Nishiyori H., Nomura K., Ohno M., Sakazume N., Sano H., Sasaki D.,
RA Shibata K., Shiraki T., Tagami M., Tagami Y., Waki K., Watahiki A.,
RA Muramatsu M., Hayashizaki Y.;

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<!--StartFragment-->                    RL   Submitted (APR-2004) to the EMBL/GenBank
CC   ---------------------------------------------------------------------
CC   Copyrighted by the UniProt Consortium, see http://www.uniprot.org/terms
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CC   ---------------------------------------------------------------------
DR   EMBL; AK159764; BAE35354.1; -; mRNA.
DR   EMBL; AK168402; BAE40323.1; -; mRNA.
DR   EMBL; AK146702; BAE27369.1; -; mRNA.
DR   UniGene; Mm.22515; -.
DR   MGI; MGI:104903; Gpc3.
DR   GO; GO:0005578; C:extracellular matrix (sensu Metazoa); RCA.
DR   GO; GO:0005615; C:extracellular space; RCA.
DR   GO; GO:0008285; P:negative regulation of cell proliferation; IGI.
DR   GO; GO:0045926; P:negative regulation of growth; IMP.
DR   GO; GO:0009887; P:organ morphogenesis; IMP.
DR   GO; GO:0030513; P:positive regulation of BMP signaling pathway; IMP.
DR   GO; GO:0001658; P:ureteric bud branching; IGI.
DR   InterPro; IPR001863; Glypican.
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human
mouse

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